

1154-92-2523

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Mathematical models of flow-mediated blood clotting, a highly localized biochemical and biophysical phenomenon, have been used to predict modifiers of both bleeding and thrombosis. These conditions are results of dysregulation of two intertwined processes of hemostasis; the function of platelets, a blood cell that circulates in the human vasculature, and the complex network of reactions known as coagulation. We have developed a temporally varying mathematical model of flow-mediated primary hemostasis in an extravascular injury. The model consists of a system of ordinary differential equations that describe platelet accumulation and blood flow through the injury. We coupled the contribution of increased resistance due to growth of the platelet aggregate to the flow through the injury using a Brinkman-Stokes-Brinkman calculation. Calculations were calibrated using an analogous partial differential equation (PDE) model and partial validation was performed using occlusion times and flow rates from microfluidic flow assays. Lastly, we investigated the effects of inhibition of soluble agonist-mediated platelet activation on two metrics of bleeding: occlusion times of the injury channel and the flow rates through the platelet aggregate. (Received September 17, 2019)